

## REFERENCES

1. Alpert JS, Thygesen K, Antman E, Bassand JP. Myocardial infarction redefined—A consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction. *J Am Coll Cardiol* 2000;36:959–69.
2. Januzzi JL, Lewandrowski K, MacGillivray TE, et al. A comparison of cardiac troponin T and creatine kinase-MB for patient evaluation after cardiac surgery. *J Am Coll Cardiol* 2002;39:1518–23.
3. Bennetts JS, Baker RA, Ross IK, et al. Assessment of myocardial injury by troponin T in off-pump coronary artery grafting and conventional coronary artery graft surgery. *Aust N Z J Surg* 2002;72:105–9.
4. Kathiresan S, MacGillivray TE, Lewandrowski K, et al. Off-pump coronary bypass grafting is associated with less myocardial injury than coronary bypass surgery with cardiopulmonary bypass. *Heart Surg Forum* 2003. In press.

## Quantitative Relationship Between Severity of Pulmonary Hypertension and LV Diastolic Function Has Been Established

We read with interest the article entitled “Correlation of Left Ventricular Filling Characteristics With Right Ventricular Overload and Pulmonary Artery Pressure in Chronic Thromboembolic Pulmonary Hypertension” published in the July 17, 2002, issue of the *Journal* (1). Dr. Mahmud and his colleagues identified an impaired relaxation pattern of left ventricular (LV) filling in 39 patients with chronic thromboembolic pulmonary hypertension (CTPH), with a mean pulmonary artery pressure  $>30$  mm Hg, which normalized after successful pulmonary thromboendarterectomy. The objective (e.g., finding a quantitative relationship between the right ventricular [RV] pressure overload and the type of LV diastolic dysfunction) and some of the results of this study (only patients with severe pulmonary hypertension have an altered LV filling) resemble those published by our group in 2001 (2). We were disappointed by the investigators’ claim in the Objectives section of their Abstract that “a quantitative relationship between RV pressure overload and LV diastolic function has not been established.”

We examined by Doppler-echocardiography 120 patients with chronic pulmonary hypertension (of whom 12 patients had CTPH), and we found that only in patients with a systolic pulmonary artery pressure (SPAP)  $\geq 60$  mm Hg is LV diastolic filling altered in the form of impaired relaxation pattern. In addition, we also found that the late systolic and early diastolic interventricular septum flattening occurs in 70% of patients with SPAP  $\geq 60$  mm Hg and only in 6% of those with SPAP  $< 60$  mm Hg. Therefore, a quantitative relationship between the severity of pulmonary hypertension and LV diastolic function was already established.

We would like to add that, in addition to abnormal geometrical configuration and motion of interventricular septum mentioned by Dr. Mahmud, other possible mechanisms of LV diastolic dysfunction in patients with severe pulmonary hypertension might be related to the presence of some degree of LV interstitial edema, which increases the LV wall stiffness and alters its normal diastolic filling (3) and the diastolic asynchrony found in the apical and lateral walls (4).

Finally, we believe that the findings of both reports complement each other and enlarge our understanding of the mechanisms of dyspnea in patients with severe pulmonary hypertension.

## Eddy Barasch, MD

St. Francis Hospital  
The Heart Center  
Department of Research and Education  
100 Port Washington Boulevard  
Roslyn, New York 11576  
E-mail: Eddy.Barasch@chsli.org

## Ali Moustapha, MD

## Vinod Kaushik, MD

## Susana Diaz, BS

## Seung-Ho Kang, PhD

The University of Texas–Houston Medical School and  
Memorial–Hermann Hospital  
Houston, Texas

doi:10.1016/S0735-1097(02)03015-2

## REFERENCES

1. Mahmud E, Raisinghani A, Hassankhani A, et al. Correlation of left ventricular filling characteristics with right ventricular overload and pulmonary artery pressure in chronic thromboembolic pulmonary hypertension. *J Am Coll Cardiol* 2002;40:318–24.
2. Moustapha A, Kaushik V, Diaz S, Kang S-H, Barasch E. Echocardiographic evaluation of left-ventricular diastolic function in patients with chronic pulmonary hypertension. *Cardiology* 2001;95:96–100.
3. Davis KL, Mehlhorn U, Laine GA, Allen SJ. Myocardial edema, left ventricular function and pulmonary hypertension. *J Appl Physiol* 1995;78:132–7.
4. Bhargava V, Sunnerhagen KS. Left ventricular asynchrony in patients with pulmonary hypertension. *J Appl Physiol* 1990;69:517–22.

## REPLY

We would like to respond to the comments by Barasch et al. regarding our study “Correlation of Left Ventricular Filling Characteristics With Right Ventricular Overload and Pulmonary Artery Pressure in Chronic Thromboembolic Pulmonary Hypertension” recently published in the *Journal* (1). The study by Moustapha, Barasch, and colleagues (2) was published only a short time before we submitted our manuscript and so, regrettably, was not included in our references.

The objective of our study was to define a quantitative relationship between right ventricular (RV) pressure overload and left ventricular (LV) diastolic filling characteristics, measuring both as continuous variables (i.e., mean pulmonary artery pressure and E/A ratio). Furthermore, we only studied patients with chronic thromboembolic pulmonary hypertension (CTEPH) who were undergoing pulmonary thromboendarterectomy (PTE) so that we could assess the change in LV diastolic filling with resolution of an RV pressure overload state.

In the study by Moustapha et al. (2), the degree of pulmonary hypertension was defined only as a dichotomous variable (mild-moderate vs. severe). Pulmonary artery (PA) pressure was estimated by Doppler-echocardiography, not measured directly as in our study. Moustapha and colleagues reported that the group of patients with severe (systolic PA pressure  $>60$  mm Hg) had lower E/A ratios than did the group with mild-moderate pulmonary hypertension (systolic PA pressure 40 to 60 mm Hg) ( $0.96 \pm 0.37$  vs.  $1.34 \pm 0.54$ , respectively [ $p < 0.05$ ]). This relationship of low E/A ratio and severe pulmonary hypertension with associated interventricular septal distortion is, however, well recognized (3,4).